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Editorial

'There is nothing new except what is forgotten.'
—attributed to Mademoiselle Bertin, milliner to Marie Antoinette [Circa 1785]

Virchow, in his book *Cellularpathologie*, tells us that every era of medical advancement is heralded by the rediscovery of anatomy, so it is perhaps not a coincidence that the rising interest in methods of treatment of cartilage disease should coincide with the recent advances in arthroscopy and imaging. The rapid improvements made in Magnetic Resonance Imaging (MRI) have led to the possibility of in-vivo examination of human anatomy in ever increasing detail. For the orthopedist and rheumatologist, the possibility of a clear, non-invasive look at the articular cartilage and other intra-articular structures is indeed exciting. However, the ability 'to see' (i.e., to see imaginatively) what the MRI has to show us depends on our physiologic understanding of normal gross and microscopic anatomy both at various ages and in morbid states. Unfortunately, as far as articular cartilage is concerned, the number of investigators who have interested themselves in the topic has been limited and the available information is conflicting.

Form follows function, not only in suspension bridges and modernist buildings, but also in the extracellular matrices of the connective tissues of plants and animals. The extracellular matrix of articular cartilage consists, for the most part, of a hydrated proteoglycan gel that is encased within a three-dimensional network of collagen fibrils. The collagen fiber network resists the tensile loads generated both during loading of the joint and by the swelling pressure of the hydrophilic proteoglycan gel entrapped by the network.

Hultzkranz in 1898[1] was one of the early investigators to demonstrate an organized fibrous structure in articular cartilage, work which was to be expanded by Benninghof and resulted in his classic papers in 1925.[2,3] Studies undertaken at Imperial College, London in the mid-1960s showed contours of stiffness on the articular surface of the femoral head which could be related to proteoglycan and water distribution and which were thought to reflect the loading history of the various parts of the joint.[4] That there is a heterogeneity in the gross appearance of the articular cartilage of a joint was reported by Bennett, Waine and Bauer[5] in their monograph on age changes in the human knee joint where they noted 'degenerative change' of the tibial plateau in all individuals over 16 years of age. These changes were especially notable in the lateral tibial plateau in the area not covered by the meniscus. Similar age-related degenerative changes have also been shown in other joints.[6] ('Degenerative changes' may be the result of injury in the form of repetitive stress. But they may also result from disuse or aging. In some tissues we know which is responsible, in others we don't.)

Electron Microscope (E.M.) studies of the architectural arrangement of collagen fiber in cartilage are not easy because of the difficulty in maintaining orientation with such tiny blocks of tissue. However, when orientation of the blocks has been achieved then it is clear that an arrangement exists.[7] Scanning electron microscope studies have suggested a lamellar or leaf-like layered arrangement of the collagen fibers which follows the arrangement of the split lines at the articular surface.[8] Presumably within the lamellae the collagen fibers are also organized in a preferred direction just as they are in the lamellae of mature trabecular and cortical bone, but at present this is not clear.

E.M. studies have also shown a quite different arrangement, as well as diameter, of collagen fibers in different areas of the joint. The diameter of the collagen fibrils varies from the surface layers where in general the fibers are of uniform thickness and measure approximately 34 nm, with finer fibers <10 nm, to the deeper cartilage where the fibers are between 70 and 100 nm.[9] However, collagen fiber diameter and distribution vary not only in the various horizontal layers from the articular surface to the tidemark but also in different anatomic locations in the joint. For example, in the mid and deep zone of the cartilage of the tibial plateau in those areas not covered by the menisci, the collagen fibers have been shown to be bundled, parallel and of a more or less uniform diameter.[10] Like the differences in proteoglycan distribution within the cartilage, these differences too have been attributed to patterns of loading and use.

The study in this issue of the journal by John Foster and his colleagues confirms the findings of previous workers that high resolution MRI reveals 'a heterogeneous deep lamina that consists of alternating hyperintense and hypointense bands perpendicular to the subchondral bone'.[11] They have correlated these findings with microscopic examination of histologic sections. Unfortunately, the joint that they used for their studies was the knee joint, the joint which shows the earliest degenerative changes and ultimately the greatest extent of degenerative changes. (Perhaps the ankle joint, of a young individual, which shows little or no propensity for degeneration or the development of osteoarthritis would have been a most suitable subject for the study of normal cartilage.)[12] However, the questions posed by morphology are never meaningless and as interest grows in ever more sophisticated therapeutics for cartilage damage, the concomitant need to monitor its efficacy, will force clinicians to rediscover anatomy for themselves and ask more searching questions concerning the meaning of these exciting new imaging modalities.

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